Electrolyte disorders in the critically ill

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Abstract

Electrolyte disorders are extremely common in the critically ill patient. Competent analysis and management of these is essential in providing quality intensive care. This article provides a review of and guide to aetiology, analysis, and management of the major electrolytes in the critically ill.

Keywords Calcium; chloride; critically ill; dysnatraemia; electrolytes; fluid; magnesium; phosphate; potassium; sodium

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 2A05, 2C01, 3C00

Learning objectives

After reading this article readers should be able to:

- recognize the multiple aetiologies of electrolyte abnormalities of the critically ill
- identify the signs and symptoms of the various electrolyte abnormalities
- prescribe appropriate management of abnormalities, in particular the management of different types of dysnatraemia

Introduction

Electrolyte disorders are extremely common in the critically ill patient. Competent analysis and management of these is essential in providing quality intensive care. Electrolyte disorders represent:

- aids to the diagnosis of the nature of the illness
- markers of disease severity and prognosis
- indications of total body deficit or excess requiring specific management.

Many electrolyte disturbances can be managed simply; deficits by increasing intake and excess by reducing intake or encouraging loss but generalizing this approach to all electrolyte and metabolic disturbances is over-simplistic. Fundamental concepts in understanding electrolyte disorders include the following.

- Serum (or plasma) levels do not always reflect total body stores of the electrolyte. For example potassium, a principally intracellular ion, is in a total body deficit in diabetic ketoacidosis (DKA) yet serum levels are elevated. Simplistic interpretation would not identify the total body deficit requiring ongoing replacement.
- An electrolyte abnormality reflects an underlying pathological process that may require definitive treatment.
- 'Correction' of a specific electrolyte abnormality may not improve a patient's condition, and may even worsen their outcome, or mask the problem.
- Established recipes for electrolyte replacement to correct abnormalities serve as a starting point, but cannot replace repeated clinical examination and serial measurement of electrolytes.
- All electrolytes are strong ions or weak acids and electrolyte disturbances may alter the acid–base status and vice-versa. This can be assessed with blood gas data.

Specific electrolytes

The normal values and effects of deficit and excess of common electrolytes are listed in Table 1.

Sodium

Thirst, vasopressin, and the kidneys control serum levels of the major extracellular cation, sodium. The prevalence of dysnatraemias in the intensive care unit (ICU) approaches 30%. They are an independent risk factor for poor prognosis on admission and during ICU stay and, not surprisingly, are incorporated into severity scoring systems such as APACHE II. Sodium has osmotic and electrostatic activity so measured hypo- or hypernatraemia needs to be assessed in correlation with the patient's volume status and serum and urinary osmolality to determine the likely cause and appropriate method of correction.

Hypernatraemia

Aetiology: in the outpatient population hypernatraemia is rarely caused by excess sodium gain, and is usually the result of water deficit relative to total body sodium. In the ICU population however, excess total body sodium due to iatrogenic loading of hypertonic fluid is not uncommon. Multiple factors lead to renal loss of hypotonic fluid in critically ill patients, which may then be replaced with a comparatively hypertonic fluid such as 0.9% saline. Examples of hypotonic fluid loss include sustained diarrhoea, vomiting or nasogastric losses, excessive sweating and central or nephrogenic diabetes insipidus. Sustained hypernatraemia can only occur when access to free water is restricted or when the thirst mechanism is absent – a common situation in ICU.

Management: as sodium is the major extracellular cation, in hypernatraemia there will be a fluid shift from intracellular to extracellular. The brain compensates to this by retaining other solutes to restore cell volume, although the hypertonic state remains. When correction commences it takes several days for

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Normal physiologic ranges of the common electrolytes and effects of deficit or excess

Electrolyte	Normal values (mmol/litre)	Effect of excess	Effect of deficit
Cations			
Sodium	136—145	Cerebral vascular rupture, haemorrhage death	Headache, lethargy, irritability, spasticity, seizures and coma, demyelination syndromes if corrected too quickly
Potassium	3.5—5.0	Peaked t-waves, can proceed to widened QRS, heart block, bradycardia, cardiac arrest, muscle weakness, paraesthesia, loss of tendon reflexes, ileus, constipation, and palpitations	Depressed ST segments, biphasic t-waves, prominent u-waves → tachyarrhythmias. Muscle weakness, paraesthesia, loss of tendon reflexes, ileus, constipation, and palpitations
Calcium	Total 2.10—2.60, ionized: 1.10—1.35	Neurological (headache, fatigue, apathy, confusion), gastrointestinal (pain, constipation, vomiting), renal (polyuria, nephrolithiasis, renal failure) cardiovascular (arrhythmia's, short QT interval and atrioventricular or bundle branch block) and skeletal (pain, arthralgia)	Tetany, paraesthesia, mental changes, areflexia, decrease in cardiac output with hypotension, dehydration via hypercalcaemic nephrogenic diabetes insipidus
Magnesium	0.6—1.2	Tetany and arrhythmias, atrial dysrhythmias, reduced cardiac output and death	Muscle weakness, decreased reflexes, hypotension bradycardia somnolence coma. Where causes include gastrointestinal disorders (malabsorption, diarrhoea, nasogastric losses, pancreatitis, and short bowel), endocrine disorders, renal losses and drugs
Anions			
Chloride	95—105	Unknown-/related to associated abnormality	Unknown-/related to associated abnormality
Phosphate	0.8—1.5	Symptoms of acute hypocalcaemia, acute tubular necrosis, ectopic calcification	Below 0.32 mmol/litre respiratory muscle dysfunction, left shift of oxyhaemoglobin dissociation curve, myocardial dysfunction, arrhythmia's, muscle weakness, insulin resistance, neuropathy, seizures, coma, haemolytic anaemia
Zinc	10.7—22.9 μmol/litre (serum) 13.8—22.9 μmol/litre (plasma)	Sparse hair, easily pluckable hair, alteration of taste; reddish dermatitis around nose, mouth and groin; hair loss, poor wound healing	Copper deficiency resulting in bone marrow abnormalities

these accumulated solutes to disperse. If large volumes of hypotonic fluid are delivered rapidly, the resultant cerebral oedema can lead to irreversible brain damage.

For this reason, correction by less than 0.6 mmol/litre/hour or 10—15 mmol/litre in a 24-hour period is recommended. Hypotonic fluid such as pure water, 5% dextrose, or 0.45% saline in the lowest volume required to correct the hypertonicty should be used.

Hyponatraemia

Aetiology: hyponatraemia can occur in the setting of low, normal, or high total body water as outlined in Table 2. It can also occur in variable states of tonicity and osmolality depending on the presence of other solutes. For example, in hyperglycaemia the excess osmotic load of glucose holds water in the extracellular space, causing a hyponatraemia that is hyperosmolar and hypertonic.

Differentiating the cause of hyponatraemia requires clinical examination of fluid status along with simple investigations to assess serum and urine sodium and osmolality.

Hypervolaemic hyponatraemia is most often related to impaired water excretion by the kidneys. Hypovolaemic hyponatraemia is most often due to renal or extrarenal concurrent sodium and water loss.

Clinical features: (Table 1) are seen when sodium derangement (hyper or hypo) is severe and/or occurs rapidly. Signs and symptoms relate predominantly to central nervous system (CNS) dysfunction. These are not usually seen until serum sodium falls below 125 mmol/litre but severity is also related to the speed of development. Acute hypotonic hyponatraemia is most dangerous as the entry of water into brain cells results in cerebral oedema and risk of tentorial herniation.

Management: in mild hypovolaemic hyponatraemia isotonic saline (0.9% saline) is usually sufficient to correct serum sodium. In mild hypervolaemic cases fluid restriction may be appropriate.

Convulsions, unconsciousness, self-induced water intoxication, and hyponatraemia associated with intracranial pathology are medical emergencies that demand prompt and definitive